Genetics and Molecular Research

TNFR1-383 A>C polymorphism association with clinical manifestations in primary Sjögren's syndrome patients

A.L. Fletes-Rayas[1], C.A. Palafox-Sánchez[2], J.F. Muñoz-Valle[2], G. Orozco-Barocio[3], R.E. Navarro-Hernández[4] and E. Oregon-Romero[2]

[1]Doctorado en Genética Humana, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, México
[2]Instituto de Investigación en Ciencias Biomédicas, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, México
[3]Hospital General de Occidente, Secretaría de Salud Jalisco, Zapopan, Jalisco, México
[4]Instituto de Investigación en Reumatología y del Sistema Musculo Esquelético, Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, México

Corresponding author: E. Oregon-Romero
E-mail: oregon_edith@hotmail.com

Genet. Mol. Res. 15 (2): gmr.15024177
Received December 9, 2015
Accepted March 9, 2016
Published June 17, 2016
DOI http://dx.doi.org/10.4238/gmr.15024177

ABSTRACT. Primary Sjögren's syndrome is an autoimmune disease affecting the function of exocrine glands. Tumor necrosis factor receptor-1 (TNFR1) is involved in apoptosis through extrinsic pathway initiation. The level of soluble TNFR1 is reported increased in rheumatoid arthritis, systemic lupus erythematosus, and primary Sjögren's syndrome patients. The *TNFR1* gene contains a polymorphism that replaced an adenine with a cytosine at the -383 in promoter region position. The *TNFR1*-383 A>C polymorphism has been associated with rheumatic diseases. We examined the association between the *TNFR1*-383 A>C polymorphism and TNFR1 soluble (sTNFR1) levels and

laboratory and clinical characteristics in primary Sjögren's syndrome patients. Eighty-two patients with primary Sjögren's syndrome classified using the American-European criteria and 84 healthy subjects were studied. Sjögren's Syndrome Disease Activity Index (SSDAI) and Sjögren's Syndrome Disease Damage Index were performed for all patients. Genotypic and allelic frequencies were similar in both groups (P = 0.317 and P = 0.329, respectively). sTNFR1 levels were similar in patients and healthy subjects (P = 0.051). High levels of C-reactive protein (P = 0.045) and rheumatoid factor (P = 0.040) in patients with the A>C genotype were observed. In these patients, the SSDAI score was higher than in A>A genotype carriers (P = 0.045). This is the first study that to examine the *TNFR1*-383 A>C polymorphism in primary Sjögren's syndrome patients. Clinical parameters and SSDAI index were associated in A>C genotype carriers. However, further studies with a larger sample are necessary to verify the association between primary Sjögren's syndrome and the *TNFR1*-383 A>C polymorphism.

Key words: Primary Sjögren's syndrome; *TNFR1*-383 A>C polymorphism; Laboratory and clinical characteristics; sTNFR1